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African Americans and Caucasians Following Radical
Prostatectomy

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FOREWORD

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Monami Banerjee 12/08/99
PI - Signature Date

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INTRODUCTION

Prostate cancer is currently the most common malignant neoplasm and the second leading cause of cancer-specific death among males in the United States. In 1998, 184,500 new patients were estimated to be diagnosed with prostate cancer (PC) and 39,200 deaths were attributed to this cancer¹⁻². In addition to the problems posed by the sheer numbers of individuals affected, statistics indicate that \$1.5 billion are spent for direct medical expenses and an additional \$2.5 billion for indirect costs for the management of prostate cancer³. African American men have higher incidence of prostate cancer than Caucasian men², and African American men in the metropolitan Detroit area have one of the highest incidence rates in the world. Mortality from prostate cancer is two to three times greater among African American men between the ages of 40 and 70 years than among similarly aged Caucasian men². Even though age-adjusted mortality rate is declining in prostate cancer, the decrease is greater among Caucasian compared to African American men, further increasing the disproportionality in death rates between the two ethnic groups. Outcome data of men treated with radical prostatectomy for clinically localized prostate cancer also demonstrate more advanced tumors at diagnosis, and for each pathologic stage, African American men have higher recurrence rates than Caucasian men. Given the differential disease outcome in the two races, it is conceivable that race may have an effect on the rate of progression in patients that experience biochemical recurrence following radical prostatectomy. Current studies in the literature are based on survival analyses of time to (biochemical) recurrence data, thus focusing only on the time from surgery to the time when prostate-specific antigen (PSA) measurement exceeds a threshold (0.4 ng/ml.), without specifically modeling how this threshold was attained. The proposed study will model follow-up serial PSAs to characterize the pattern of progression in patients who suffer biochemical recurrence, and will produce rates of progression for African Americans and Caucasians. The results will allow us to establish if African Americans indeed require earlier therapeutic intervention in order to reduce the disproportionality in survival outcomes between the two ethnic groups.

BODY

This section describes the research accomplishment associated with each task outlined in the approved Statement of Work.

TASK : Meet with collaborating established investigator and urologist for clinical data interpretations.

The PI met with Drs. Severson and Powell on 2/12/99 and 3/29/99 to discuss analytic strategies for the project. In particular, a list of clinicopathological variables to be included in the analysis was generated, along with range and unit of measurement for each variable. Also discussed were exclusion criteria for the study. Dr. Powell provided an overview of the radical prostatectomy database along with demographics for this cohort, and interpretations of the clinicopathological parameters available in the database. Discussion also ensued regarding data acquisition specifics, *e.g.* timeline, required format for statistical analysis, confidentiality issues, *etc.*

TASK : Review of current radical prostatectomy database for serial PSA measurements and demographic and clinicopathologic parameters.

Between January 1991 and December 1996, 1080 consecutive men underwent radical retropubic prostatectomy for clinically localized prostate cancer at the Wayne State University and Karmanos Cancer Institute affiliated Harper Hospital. The mean \pm standard deviation age of this cohort was 62.4 \pm 6.6 years (range 39 to 77 years). Fifty-seven percent of these men were Caucasians, 39% were African Americans, and the remaining 4% comprised of other ethnicities (Asian, Hispanic, and international patients). Clinical and pathological data for all patients were prospectively entered into the radical prostatectomy database. Available parameters include preoperative serum prostate specific antigen (PSA) level (in ng/ml.), clinical and pathological stage, Gleason grade of the needle biopsy and radical prostatectomy specimen, presence of extraprostatic disease, seminal vesicle invasion and/or lymph node metastasis. Postoperative follow-up consisted of serum PSA level determinations (along with digital rectal examination) every 3 months for the first two years, and semiannually thereafter. Median follow-up for this cohort was 39 months. Biochemical recurrence of prostate cancer is defined as a progressive or sustained elevation of PSA; and is based on an elevated PSA level of ≥ 0.4 ng/ml. Of the 1080 patients in the entire radical prostatectomy database, 22.3% (n=241) had biochemical recurrence on or before January, 1999; among which 44.4% (n=107) recurred within 1 year of surgery, and 63.9% (n=154) recurred within the first 2 years of surgery.

Inclusion into our study required

- Evidence of biochemical recurrence (as documented by an elevated PSA level of ≥ 0.4 ng/ml) on or before January, 1999.
- An undetectable serum PSA following surgery.
- No pre-operative hormonal or radiation therapy.
- No postoperative hormonal or radiation therapy until documented evidence of biochemical recurrence.
- Race African American or Caucasian.

Seventy-seven of the men fit the above criteria and constitute the cohort of men for statistical analysis. Patient profiles are characterized by serial PSA measurements recorded at consecutive follow-ups.

Most studies so far have only focused on the time to biochemical recurrence, *i.e.* the time from surgery to the time when the PSA exceeded the threshold (0.4 ng/ml.), without specifically modeling how this threshold was attained. The current study models intermediate serial PSA measurements to investigate if African American men who experience biochemical recurrence after radical prostatectomy exhibit a differential rate of progression compared to their Caucasian counterparts. Thus, for each of the eligible patients, we look at the individual profiles **till the time of first recurrence**. As an illustration, consider the patient profile in Figure 1.

This patient had his first follow-up PSA done 8.8 months after surgery and had an undetectable PSA (0.05 ng/ml.) at that point of time. His PSA remained undetectable till about 33.5 months after surgery (he had 4 serial PSAs during that time interval). At 45.7 months post-surgery his PSA rose to 0.1 ng/ml., followed by 2 consecutive PSA values of 0.21 ng/ml. at 47.5 and 60.5 months post-surgery. After about 9 months following this measurement, his PSA rose to 0.33 ng/ml. Subsequently, at 76 months post-surgery his PSA increased to 0.56 ng/ml., at which point he was declared to have biochemical recurrence and was put on hormonal therapy. Thus, this patient had 10 serial PSA measurements from his time of surgery to his time of first recurrence, and the current study models this entire profile based on the available 10 PSAs.

For our analysis cohort of 77 men, Table 1 shows the frequency distribution of the available number of serial PSAs. Thus, 71% of the patients in our study cohort had at least 4 serial PSA measurements. The distribution according to race was as follows : out of the 77 men, 46 (59.7%) were Caucasians and 31 (40.3%) were African Americans. The mean (\pm standard deviation) age at surgery of men in our study cohort was 65 (\pm 6.6) years and 10.4% of the men were less than or equal to 55 years of age. Table 2 shows the frequency distribution according to preoperative PSA. Only 5% of the patients in our study cohort had preoperative PSAs less than or equal to 4 ng/ml., 39% had PSAs between 4.1-10 ng/ml., 25.9% had PSAs between 10.1-20 ng/ml., and 29.9% had PSAs above 20 ng/ml. Clinical stage of these patients ranged from T1c to T2b, with 10.4% of the men presenting with clinical stage T1c disease, 59.7% presenting with T2a disease, and 29.9% presenting with T2b disease. Pathological stage was classified as : organ confined (CON), positive surgical margin (SM), extraprostatic extension (EPE), seminal vesicle invasion (SVI) and lymph node metastasis (LN). Approximately 11.7% of the patients in our study cohort had organ confined disease, 18.2% had positive surgical margin, 31.1% had extraprostatic extension, 27.3% had seminal vesicle invasion, and 11.7% had lymph node metastasis. Gleason grade of the needle biopsy as well as radical prostatectomy specimen were scored on a scale of 2-10. Previous studies⁴ have demonstrated that patients with Gleason score 7 have disease outcomes that are intermediate to patients with Gleason ≤ 6 and Gleason ≥ 8 . Based on these categorizations, the distribution of biopsy Gleason scores in our study cohort was as follows : 49.4% had Gleason ≤ 6 , 41.6% had Gleason =7, and 9% had Gleason ≥ 8 . On an average, there was an upgrading in the radical prostatectomy specimen with only 9.1% having prostatectomy Gleason ≤ 6 , 59.7% having Gleason =7, and 31.2% having Gleason ≥ 8 .

TASK : Review existing statistical literature on random effects models for repeated measurements data.

Longitudinal or repeated measures analysis is most appropriate for the investigation of individual changes of PSA over time and for the study of effects of race and other factors likely to influence change. In principle, continuous data from designed longitudinal experiments can often be analyzed with classical multivariate regression techniques. However, such methods impose model assumptions that are usually not met in observational studies, since the circumstances under which the measurements are collected cannot always fully be controlled. Patients in our study had different number of follow-up PSAs, at different periods of time, and the intervals between successive PSA measurements were different as well. This unbalancedness in the study design makes classical multivariate regression techniques infeasible. Secondly, in a truly multivariate set of outcomes, the variance-covariance matrix is usually unstructured, in contrast to longitudinal data, where the correlations of the repeated measurements are likely to be smaller for observations that are further apart in time.

One useful alternative for modelling such data is the linear random effects model^{5,6,7,8,9}, in which the repeated measurements are modeled using a linear regression model, with parameters which are allowed to vary over patients, and which are therefore called random-effects or patient-specific regression coefficients. Since there is too little data on a single patient to estimate its regression parameters, and to avoid theoretical obstacles, one often assumes that the random effects are independently and identically distributed random variables. Their distribution is referred to as the mixing distribution. Furthermore, since the patient-specific regression parameters reflect the natural heterogeneity in the population and because they can also be interpreted as the deviation of a specific patient's profile from the overall population profile, they are usually assumed to follow a Gaussian distribution. Their mean then reflects the average profile in the population, and is therefore called the vector of fixed effects. The assumption of a Gaussian mixture is not only intuitive, it is also

mathematically convenient because it implies that both the marginal distribution of the data and the posterior distribution of the random effects are Gaussian, which considerably simplifies the estimation procedure.

The above class of models is more commonly referred to as linear mixed (-effects) models since it involves both fixed effects and random effects. The fixed effects are regression parameters which are assumed to be the same for all subjects, while the random effects are subject-specific regression coefficients. Inference for the linear mixed model is based on maximum likelihood and restricted maximum likelihood. Laird and Ware⁵ present a two-stage formulation, which is particularly useful to provide intuition into the linear mixed model. The first stage summarizes individual profiles in terms of their proper regression coefficients. In the second stage, regression models are fitted to these subject-specific quantities, leading to population averaged parameters such as race or age effects.

In order to obtain a clear picture of how random effects might be operating it is important to first find a parsimonious description of the random-effects structure. Technically, this involves testing for variance components. If belief is granted to the selected random effects structure, it is sometimes of interest to estimate the patient-specific profiles. Since this involves the estimation of the random effects, it is most natural to adopt Bayesian techniques.

A linear mixed effects model requires not only the specification of an adequate mean structure, but also the description of a covariance structure. Misspecifying the mean structure will affect the covariance structure and vice versa. Parametric models for covariance structure are particularly useful for data in which the measurements on different patients are not made at a common set of times (as in our case with the serial PSA data).

TASK : Review the appropriate analytic modules or codes within statistical software programs SAS and S-plus.

The dissemination of the MIXED procedure in SAS and the *lme* function in S-Plus has provided the opportunity to fit the class of linear mixed effects models for routine use. The SAS procedure MIXED was used for this research since the PI is more conversant with SAS, and data manipulation is easier in SAS. Also, the MIXED procedure allows greater flexibility in the modeling of covariance structures, which was an important consideration for the serial PSA data.

Within SAS, the PROC MIXED¹⁰ statement calls the procedure MIXED. The procedure requires that the data set is structured such that each record corresponds to the measurements available for a subject at only one point in time. The option "method = " within this statement specifies the estimation method. Restricted maximum likelihood is the default method, although maximum likelihood and minimum variance quadratic unbiased estimation are available options. In principle, the SAS procedure MIXED uses distinct statements to specify random-effects models (the RANDOM statement) and marginal models (the REPEATED statement). The RANDOM statement defines the matrices containing the covariates with subject-specific regression coefficients. The "subject = " option within the RANDOM statement is used to identify the individual patients in the data set. All records with the same value for the patient identifier are assumed to be from the same patient, whereas records with different values for the patient identifier are assumed to contain independent data. The "type = " option specifies the covariance structure for the random effects. Although many structures are available, in longitudinal data analysis, one usually specifies "type = UN" which does not assume the random-effects covariance matrix to be of any specific form. The REPEATED statement is used to specify the correlation matrix for repeated measurements within a subject. The

repeated effects define the ordering of the repeated measurements within each subject. These effects must be classification variables. Usually, one will specify only one repeated effect. Its levels should then be different for each observation within a subject. If not, PROC MIXED constructs identical rows in the correlation matrix corresponding to the observations with the same level, yielding a singular correlation matrix and an infinite likelihood. The options for the REPEATED statement are similar to those for the RANDOM statement. The "type =" option specifies the covariance structure for the error components within a subject. Structures available include compound symmetry (CS), first-order autoregressive (AR(1)), toeplitz (TOEP), spatial power law (SP(POW)), unstructured (UN), *etc.* For the serial PSA data, we used the SP(POW) structure for the repeated measurements within each patient. This is a time-series type covariance structure which provides a direct generalization of the AR(1) structure for equally spaced data. With this structure, the correlations decline as a function of time. A detailed description of all available covariance structures within SAS procedure MIXED is in Littell et. al.⁹ and the SAS/STAT documentation for PROC MIXED¹⁰. Output from SAS procedure MIXED include covariance parameter estimates, estimates for the fixed effects and corresponding F-tests, model fit information (e.g. log likelihood from the restricted maximum likelihood estimation, two model-fit criteria, namely Akaike's Information Criterion and Schwarz' Bayesian Criterion), and information on the maximum likelihood or restricted maximum likelihood iteration procedure. Further details of the SAS MIXED procedure are in references 9 and 10.

TASK : Attend sessions on *Analysis of Longitudinal Data* at the International Biometric Society Conference, December 13-18 at Cape Town, South Africa.

The conference in South Africa was scheduled from December 13-18, 1998. Unfortunately, due to administrative delays the current project did not start till April, 1999, thus precluding the PI from attending this conference. Instead, the PI attended the 1999 Spring Meeting of the International Biometric Society Eastern North American Region, held in Atlanta, Georgia from March 28-31, 1999. She attended several contributed and invited papers sessions on Longitudinal and Repeated Measures Data Analysis and chaired a session on Latent Class and Other Mixed Models. Some of the topics covered in these sessions include random effects models for reproductive hormone data, latent variable models for longitudinal data with multiple continuous endpoints, Bayesian analysis of longitudinal mortality data, and missing data in longitudinal analysis.

TASK : Perform data analyses : model serial PSA measurements using random effects models, and obtain rates of progression for African Americans and Caucasians.

Various statistical modeling strategies were adopted to study the rate of rise of serial PSA for patients experiencing biochemical recurrence following radical prostatectomy, and to compare these rates for African American versus Caucasian men. Initially, we conducted exploratory analyses of individual patient profiles as a guiding step to the more formal model building process. For patients in our study cohort, PSA values after surgery increase exponentially till the time of first recurrence. Figures 2a and 2b present some randomly selected patient profiles for the two ethnic groups. Examination of the patient profiles demonstrate

1. Unequal spacings between successive PSA measurements, *i.e.* patient follow-ups are irregular. For example, the patient marked • in Figure 2b had his first follow-up PSA test done 2.5 months after surgery, followed by a second PSA test approximately 6 months afterwards, at which point his PSA was still undetectable (0.05 ng/ml). His next PSA test was not done until about 19 months later, at which point his PSA was found to have risen to 0.4 ng/ml.
2. Wide variability across patients in the timings of follow-up. For example, in Figure 2a, the patients marked * and x generally had more regular follow-ups compared to the patient marked •, thereby enabling us to estimate the true underlying profiles of the first two patients with

better precision. For the patient marked •, the time-interval between his last undetectable PSA and his biochemical recurrence was more than 2 years (precisely, 25 months). Chances are that he actually had biochemical recurrence much earlier (since his PSA was already on the rise) and had he appeared for more regular follow-ups, this would have been detected earlier. An extreme case in point is the patient marked * in Figure 2b.

Figure 3 shows an unusual patient profile. This patient had his first follow-up PSA test done 1.4 months after surgery, followed by a second PSA test approximately 10 months afterwards, a third one 6 months afterwards, and a fourth one 5 months afterwards; till which point his PSA was well below the 0.4 ng/ml threshold, although on the rise. His next follow-up occurred almost 3.5 years later, at which point his PSA was found to have risen to 7.31 ng/ml.

Among patients in our study cohort, the distribution of age at diagnosis, preoperative PSA, clinical and pathological stage, biopsy and prostatectomy gleason did not vary significantly by race. Fifty-seven percent of the Caucasian patients and 52% of the African American patients ($p=0.67$) in our study cohort were above 65 years of age. Table 3 shows the distribution of preoperative PSA, clinical and pathological stage, biopsy and prostatectomy gleason in each racial group and the corresponding p -values from a chi-square test (p -value from exact test reported when there were sparse cells) for comparing the distribution of each clinicopathological parameter between the two races.

Mixed effects regression model was used to test the hypothesis that PSA values increase faster in African Americans compared to Caucasians. Since the pattern of PSA after surgery was to be analyzed by modeling, only patients who had four or more serial PSA values were analyzed, to ensure accurate fit. However, the effects of race estimated from both sets of models (*i.e.* models based on all 77 patients, as well as models based on the 55 patients who had four or more serial PSAs) were similar. Serial PSA values were transformed by the natural logarithm to achieve normality, which allows estimation of PSA rate of increase using the least squares regression.

Since the African Americans and Caucasians in our study cohort were similar in terms of age at diagnosis, preoperative PSA, clinical and pathological stage, biopsy and prostatectomy gleason (data presented in Table 3), no adjustment for the above covariates was incorporated in the model. Our model included random effects to account for the natural heterogeneity in transformed PSA values and the rate of change in transformed PSA within the population. This heterogeneity would be expected due to uncontrolled factors affecting when the diagnosis was made and affecting the rate of progression of prostate cancers. The fixed effects part of our model included a linear and quadratic effect of time, expressed in months since surgery, main effect of race, and interactions of race with time. The latter is used to investigate if race influences a patient's evolution over time.

For the serial PSA data, it is sensible to consider some kind of time-series covariance structure, where the correlations of the repeated measurements are assumed to be smaller for observations that are further apart in time. However, many of the time-series covariance structures available in PROC MIXED¹⁰ were inappropriate in our setting because they assume equal spacing. The compound symmetry (CS) and unstructured (UN) structures are still appropriate; however, CS assumes that the correlations remain constant, and UN is often too general. To fit a time-series type covariance structure in which the correlations decline as a function of time, we used the SP(POW) (spatial power law) structure available in PROC MIXED¹⁰. The SP(POW) structure for unequally spaced data provides a direct generalization of the AR(1) structure for equally spaced data. SP(POW) models the covariance between two measurements at times t_1 and t_2 as $\text{cov}(y_{t_1}, y_{t_2}) = \sigma^2 \rho^{|t_1-t_2|}$, where ρ is an autoregressive parameter assumed to satisfy $|\rho| < 1$ and σ^2 is an overall variance. All of the above three covariance structures (*i.e.* compound symmetry, spatial power law, and unstructured) were

fitted, and the model for final inference was selected based on Akaike's Information Criterion^{9,10} (AIC) and Schwarz' Bayesian Criterion^{9,10} (BIC). These are two model-fit criteria computed by PROC MIXED and are essentially log likelihood values penalized for the number of parameters estimated. The covariance structure with values of the criteria closest to zero is considered most desirable. Based on both the AIC and BIC, the spatial power law structure gave the best fit for the covariance model. All inferences reported for the fixed effects are therefore based on the spatial power law model for the covariance structure. Note that although we were not interested in the covariance structure in its own right, choice of a good model for the covariance structure was critical for making valid computations and inferences about fixed effects.

Based on our final model, we found highly significant linear (p-value = 0.0001) and quadratic (p-value = 0.0001) effects of time on the logarithm PSA values. This finding confirmed our earlier observation based on graphs of individual patient profiles. However, **there was no statistically significant difference in the relative rates of increase of serial PSAs between African Americans and Caucasians** (p-value = 0.73). For African Americans, the relative rate of rise of PSA after radical prostatectomy was estimated to be 0.10 ng/ml. per month, compared to 0.09 ng/ml. per month for Caucasians.

TASK : Meet with collaborating investigator to discuss research progress and to interpret data analyses results.

The PI met with Drs. Severson and Powell on 06/09/99, 09/13/99 and 10/05/99 to discuss research progress and interpret data analyses results. At the 06/09/99 meeting, the PI presented the study cohort comprising of 77 patients who fit all the inclusion criteria. At the same meeting, the PI presented patient-specific PSA profiles from the time of surgery to time of last follow-up and discussion ensued about whether to model the entire profile or the profile till the time of first recurrence. The group agreed that the first recurrence was the appropriate endpoint for the hypothesis of interest. In this meeting the PI also presented descriptive results comparing African American and Caucasian patients in the study cohort in terms of age, preoperative PSA, stage and Gleason grade distributions. At the 09/13/99 meeting, the PI presented findings from the data analyses using the mixed effects modeling approach. Discussion ensued regarding refinements of the models for serial PSA & these were incorporated by the PI in the final analyses and was subsequently presented at the 10/05/99 meeting.

TASK : Prepare manuscript for publication.

Two manuscripts detailing findings from this project are currently under preparation and are expected to be submitted by the early part of next year. Both have undergone the first set of iterations. One is being targeted for a clinical audience (to be submitted to *Cancer*), and the other will deal with methodological perspectives for serial PSA modeling (to be submitted to *Statistics in Medicine*).

TASK : Consolidate information obtained during Phase II.

This report and the two manuscripts under preparation consolidate information obtained during Phase II of the project.

TASK : Formulate research questions for DOD Idea Award Proposal based on the information obtained during Phase II.

Given the substantial evidence (that **relative rates of increase of serial PSAs following radical prostatectomy do not vary by race**) obtained from the current study, undertaking a prospective study to explore if African Americans indeed require and benefit from early therapeutic intervention no longer poses a viable next step. Also, since the award of this proposal, the PI has changed departments within the School of Medicine, and is no longer a member of the departments of Urology and Pathology. Thus, she no longer has access to new followup PSA data maintained by the department of Urology.

TASK : Submit final report summarizing results from the analyses and the conclusions obtained.

This report being submitted to the U.S. Army Medical Research and Materiel Command summarizes results from the analyses and the conclusions obtained.

KEY RESEARCH ACCOMPLISHMENTS

- Compared African American versus Caucasian patients who suffer biochemical recurrence following radical prostatectomy in terms of age at diagnosis, preoperative PSA, clinical and pathological stage, and biopsy and prostatectomy Gleason grade.
- Characterized average patient profiles based on follow-up PSAs for men who suffer biochemical recurrence following radical prostatectomy.
- Obtained relative rates of rise of serial PSAs in African American and Caucasian patients who suffer biochemical recurrence following radical prostatectomy.
- Compared relative rates of progression in African American and Caucasian patients who suffer biochemical recurrence following radical prostatectomy.

REPORTABLE OUTCOMES

1. Banerjee M., Powell I., Biswas D., and Severson R. Rates of Increase of Serial PSA in African Americans and Caucasians Following Radical Prostatectomy. (In Preparation)
2. Banerjee M., Biswas D., Powell I., and Severson R. A Mixed Effects Model for Serial PSA Data. (In Preparation).

CONCLUSIONS

This study found no significant effect of race on rate of progression for patients who suffer biochemical recurrence following radical prostatectomy for clinically localized prostate cancer. The age at diagnosis, preoperative PSA, clinical and pathological stage, biopsy and prostatectomy Gleason grade also did not vary by race in this cohort of patients.

Results from this research does not warrant the need for earlier therapeutic intervention in African Americans compared to Caucasian patients who demonstrate signs of rising PSA following radical prostatectomy for clinically localized prostate cancer.

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APPENDICES

- **List of tables (Tables 1-3).**
- **List of figures (Figures 1-3).**
- **List of personnel receiving pay from the research effort.**

Table 1 : Frequency distribution of available number of serial PSAs

# of serial PSAs	Frequency (# of patients)	Percentage
2	8	10.4
3	14	18.2
4	20	25.9
5	16	20.8
6	6	7.8
7	5	6.5
8	2	2.6
9	2	2.6
10	2	2.6
11	2	2.6
TOTAL	77	100

Table 2 : Distribution of preoperative PSAs

Preoperative PSA (ng/ml.)	Frequency (# of patients)	Percentage
0-4	4	5.2
4.1-10	30	39.0
10.1-20	20	25.9
>20	23	29.9
TOTAL	77	100

Table 3 : Distribution of Clinicopathological Parameters by Race

	RACE		
Parameter	Caucasians	African Americans	p-value
Preoperative PSA ng/ml)			
0-4	1/46 (2.1%)	3/31 (9.7%)	0.78
4.1-10	20/46 (43.5%)	10/31 (32.3%)	
>10	25/46 (54.4%)	18/31 (58.0%)	
Clinical Stage			
T1C	5/46 (10.9%)	3/31 (9.7%)	0.98
T2A	27/46 (58.7%)	19/31 (61.3%)	
T2B	14/46 (30.4%)	9/31 (29.0%)	
Pathological Stage			
Confined	8/46 (17.4%)	1/31 (3.2%)	0.67
Surgical Margin +	7/46 (15.2%)	7/31 (22.6%)	
Extraprostatic Extension	12/46 (26.1%)	12/31 (38.7%)	
Seminal Vesicle Invasion	13/46 (28.3%)	8/31 (25.8%)	
Lymph Node Metastasis	6/46 (13.0%)	3/31 (9.7%)	
Biopsy Gleason			
<= 6	22/46 (47.8%)	16/31 (51.6%)	0.85
7	20/46 (43.5%)	12/31 (38.7%)	
>= 8	4/46 (8.7%)	3/31 (9.7%)	
Prostatectomy Gleason			
<= 6	6/46 (13.0%)	1/31 (3.2%)	0.22
7	27/46 (58.7%)	19/31 (61.3%)	
>= 8	13/46 (28.3%)	11/31 (35.5%)	

Figure 1 : A sample patient profile

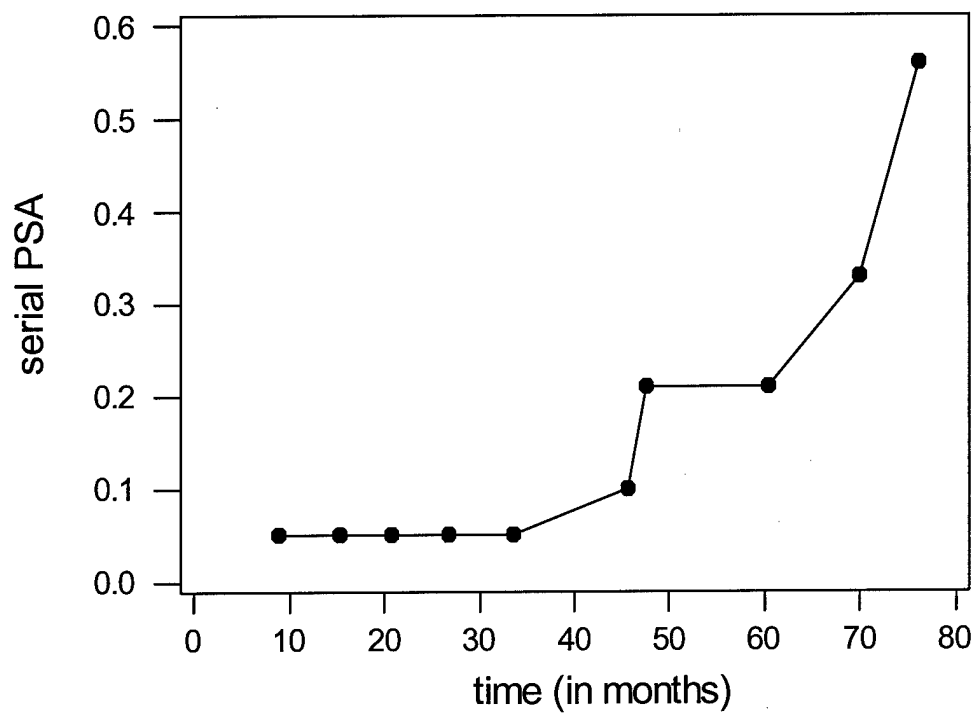


Figure 2a : Randomly selected patient profiles
Race = Caucasian

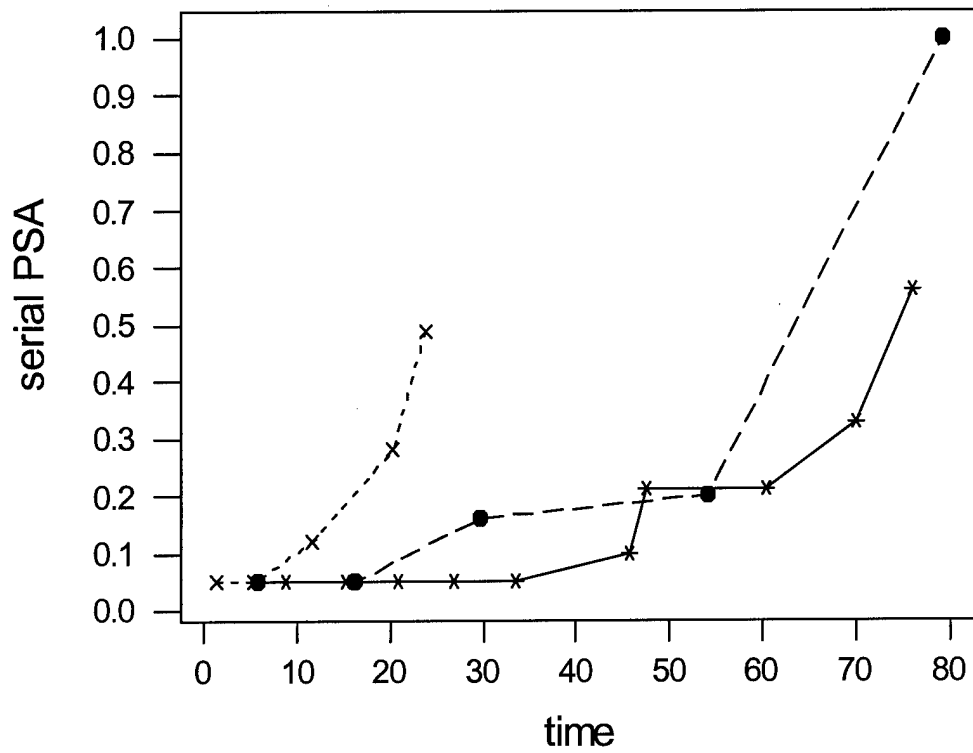


Figure 2b : Randomly selected patient profiles
Race = African American

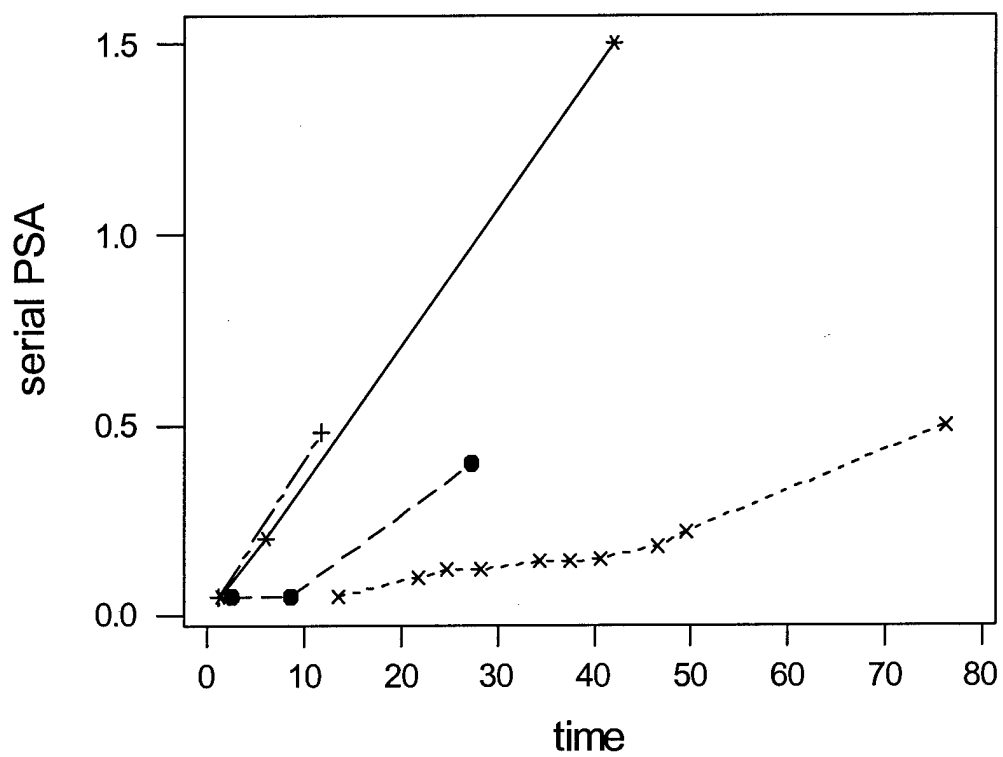
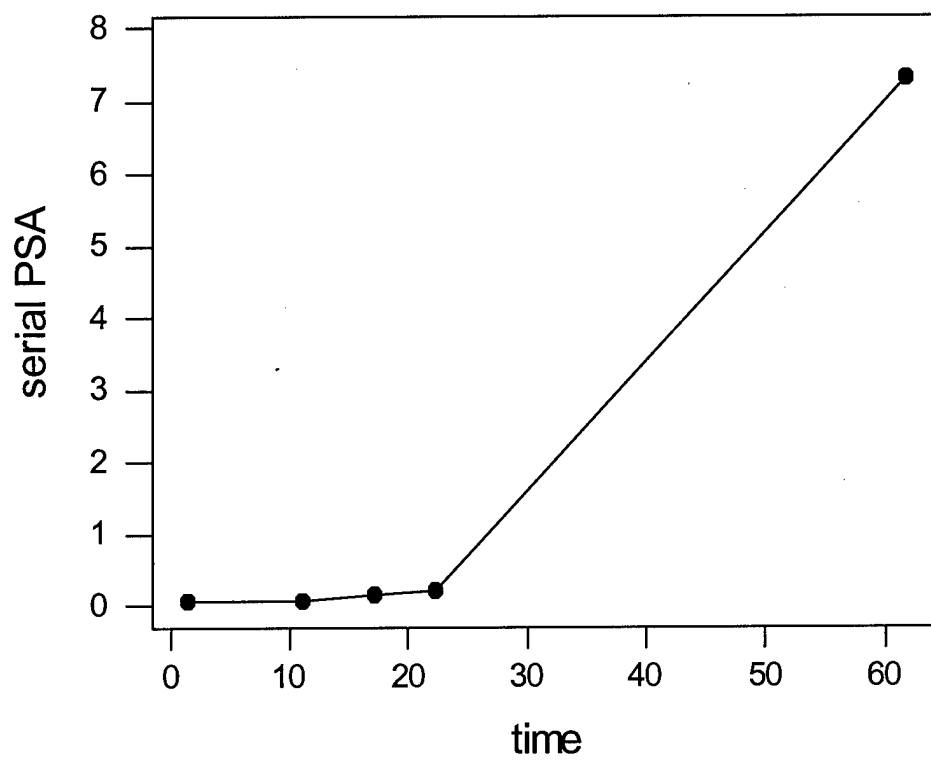


Figure 3 : An unusual patient profile



Personnel receiving pay from the research effort :

- 1. Mousumi Banerjee, Ph.D. Principal Investigator.**
- 2. Debjit Biswas, M.S. Research Assistant.**